WHAT IS CLAIMED IS:

1. A method for attracting a glial progenitor cell, or a progeny of a glial progenitor cell, to a site in a central nervous system (CNS) tissue, the method comprising the steps of:

(a) providing a compound that binds to an epidermal growth factor (EGF)/ ErbB family receptor; and

(b) administering a sufficient amount of the compound to the site to attract the glial progenitor cell or progeny thereof to the site in the central nervous system (CNS) tissue.

2. The method of claim 1, further comprising administering a sufficient amount of the compound to stimulate differentiation of the glial progenitor cell or progeny thereof.

3. The method of claim 1, wherein the compound is administered *in vivo*.

4. The method of claim 1, wherein the compound is administered by intracerebroventricular infusion.

5. The method of claim 1, wherein the compound is administered by intrastriatal infusion.

6. The method of claim 1, wherein the central nervous system (CNS) tissue is brain tissue.

7. The method of claim 6, wherein the brain tissue is adjacent to a subependymal zone.

8. The method of claim 1, wherein the central nervous system (CNS) tissue is spinal cord tissue and spinal nerve root origins.

9. The method of claim 1, wherein the compound comprises an amphiregulin (AR).

10. The method of claim 1, wherein compound comprises a betacellulin (BTC).

11. The method of claim 1, wherein the compound comprises an epiregulin (ER).

12. The method of claim 1, wherein the compound comprises a heparin-binding EGF-like growth factor (HB-EGF).

13. The method of claim 1, wherein the compound comprises a schwannoma-derived growth factor (SDGF).

14. The method of claim 1, wherein the compound comprises a myxomavirus growth factor Shope fibroma virus growth factor.

15. The method of claim 1, wherein the compound comprises a teratocarcinoma-derived growth factor-1 (TDGF-1).

16. The method of claim 1, wherein the compound comprises a transforming growth factor alpha (TGFα).

17. The method of claim 1, wherein the compound comprises a vaccinia growth factor (VGF).

18. The method of claim 1, wherein the compound comprises a heregulin.

19. The method of claim 1, wherein the compound comprises a neuregulin-3.

20. The method of claim 1, wherein the compound is administered to a tissue culture comprising a glial progenitor cell.

21. The method of claim 1, further comprising administering a second compound, wherein the second compound is capable of increasing the expression of a cell adhesion molecule or an extracellular matrix molecule.

22. The method of claim 21, wherein the second compound is transforming growth factor beta (TGF-β)

23. The method of claim 21, wherein the cell adhesion molecule is fibronectin.

24. The method of claim 21, wherein the cell adhesion molecule is laminin.

25. The method of claim 21, wherein the second compound is administered along or at the end of a desired path of migration of the glial progenitor cell or its progeny thereof.

26. The method of claim 25, wherein the compound is applied along a path between the glial precursor cell and a site toward which it is desired that the progeny migrate.

27. The method of claim 1, further comprising administering a second compound, wherein the second compound is capable of inhibiting a naturally occurring signal that would otherwise inhibit migration of the glial progenitor cell or its progeny.

28. The method of claim 1, further comprising mechanically disrupting tissue in the central nervous system (CNS), thereby directing migration of the glial progenitor cell or its progeny.

29. The method of claim 1, further comprising neurochemically blocking the activity of the central nervous system (CNS) tissue, thereby directing migration of glial progenitor cell or its progeny.

30. The method of claim 1, further comprising administering a second compound, wherein the second compound is capable of stimulating differentiation in the the glial progenitor cell or its progeny.

31. The method of claim 30, wherein the differentiation-stimulating compound comprises a retinoic acid.

32. The method of claim 30, wherein the differentiation-stimulating compound comprises a brain-derived neurotrophic factor.

33. A method for attracting a glial progenitor cell, or a progeny thereof, to a site in a central nervous system (CNS) tissue comprising administering a sufficient amount of transforming growth factor alpha (TGFα) polypeptide, or functional fragment thereof, to the site to attract the glial progenitor cell or its progeny to the site.

34. A method for ameliorating a neurological deficit in a patient, the method comprising administering to a central nervous (CNS) system tissue of the patient a compound that binds to an epidermal growth factor (EGF)/ ErbB family receptor, wherein the administration stimulates the proliferation of a glial progenitor cell or its progeny, thereby ameliorating the neurological deficit.

35. The method of claim 34, further comprising administering a sufficient amount of the compound to direct the migration of the glial progenitor cell or its progeny a desired site in the central nervous system (CNS).

36. The method of claim 34, further comprising administering a sufficient amount of a second compound to stimulate the differentiation of the glial progenitor cell or its progeny to the site.

37. The method of claim 34, wherein the neurological deficit is caused by a neurodegenerative disease, a neurotoxic injury, a disease of the spinal cord or an infection.

38. The method of claim 34, wherein the neurological deficit is caused by a developmental disorder.

39. The method of claim 34, wherein the neurological deficit is caused by an inflammatory disease.

40. The method of claim 34, wherein the neurological deficit is caused by an autoimmune disease.

41. The method of claim 34, wherein the neurological deficit is caused by a disorder affecting vision.

42. The method of claim 34, wherein the neurological deficit is caused by a disorder affecting audition.

43. The method of claim 34, wherein the neurological deficit is caused by a disorder affecting somatosensation.

44. The method of claim 34, wherein the neurological deficit is caused by a disorder affecting olfaction.

45. The method of claim 34, wherein the neurological deficit is a traumatic injury.

46. The method of claim 45, wherein the traumatic injury is an injury of the spinal cord.

47. The method of claim 34, wherein the neurological deficit is a demyelinating disease.

48. The method of claim 34, wherein the neurological deficit is Alzheimer's Disease.

49. The method of claim 34, wherein the neurological deficit is Huntington's Disease.

50. The method of claim 34, wherein the neurological deficit is Parkinson's Disease.

51. The method of claim 50, wherein administration of the compound results in differentiation of striatal cells into dopamine-producing cells.

52. The method of claim 34, wherein the neurological deficit is an ischemia.

53. The method of claim 52, wherein the ischemia is associated with a stroke.

54. A method for ameliorating a neurological deficit in a patient, the method comprising administering a first compound to a first site in a patient's central nervous system (CNS), wherein the first compound is capable of binding to an epidermal growth factor (EGF)/ErbB family receptor, with the proviso that the compound is not an EGF polypeptide, wherein the binding of the first compound to the EGR receptor results in signal transduction sufficient to stimulate the proliferation of the glial progenitor cell and its

progeny and to direct migration of the glial progenitor cell and its progeny to a second site in the central nervous system (CNS), thereby ameliorating the neurological deficit.

55. The method of claim 54, further comprising administering a second compound, wherein the second compound is capable of stimulating differentiation of the glial progenitor cell or its progeny.

56. The method of claim 55, wherein the first compound and the second compound are administered sequentially.

57. A pharmaceutical composition comprising a compound and a pharmaceutically acceptable carrier, wherein the compound comprises a first agent capable of binding to an epidermal growth factor (EGF)/ErbB family receptor and a second agent capable of stimulating the differentiation of glial progenitor cells.

58. The pharmaceutical composition of claim 57, wherein the first agent comprises a polypeptide that binds the epidermal growth factor (EGF)/ErbB family receptor.

59. The pharmaceutical composition of claim 58, wherein the polypeptide is a transforming growth factor alpha (TGFα) polypeptide, a functional variant thereof, or a biologically functional equivalent thereof.

60. The pharmaceutical composition of claim 57, wherein the second agent comprises a brain-derived neurotrophic factor polypeptide or a retinoic acid.

61. A method for ameliorating a neurological deficit, the method comprising contacting a glial progenitor cell of the patient's central nervous system (CNS) with a transforming growth factor alpha (TGFα) polypeptide, a functional variant thereof, or a biologically functional equivalent thereof, that binds to an epidermal growth factor (EGF)/ErbB family receptor expressed by the glial progenitor cell, wherein the binding of the TGFα polypeptide to the EGF receptor results in a signal transduction sufficient to stimulate

the proliferation of the glial progenitor cell and to direct migration of the progeny of the proliferating glial progenitor cell to a region of the central nervous system (CNS) in which the progeny cells can function in a manner sufficient to ameliorate the neurological deficit.

62. A method for ameliorating a neurological deficit, the method comprising contacting a glial progenitor cell of the patient's central nervous system (CNS) with a polypeptide that binds to an epidermal growth factor (EGF)/ErbB family polypeptide family receptor to which a transforming growth factor alpha (TGFα) polypeptide, a functional variant thereof, or a biologically functional equivalent thereof can bind, with the proviso that the polypeptide is not an EGF polypeptide,

wherein the binding of the polypeptide to the EGF polypeptide family receptor results in a signal transduction sufficient to stimulate the proliferation of the glial progenitor cell and to direct migration of the progeny of the glial progenitor cell to a region of the central nervous system (CNS) in which the progeny cells can function in a manner sufficient to reduce the neurological deficit.

Add a4